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### Nitric oxide function in plant abiotic stress

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**Table 2: Differential S-nitrosylation of individual proteins related to abiotic stress.**

S-nitrosylated proteins related to abiotic stress	Activity change after S-nitrosylation	Cellular localization of the protein	Reference
AHb1	ND	Cytoplasm	(Perazzolli <i>et al.</i> 2004)
APX	+	Cytoplasm	(Bai <i>et al.</i> 2011; Begara-Morales <i>et al.</i> 2012)
GAPDH ( <i>in vitro</i> only)	-	Cytoplasm	(Wawer <i>et al.</i> 2010; Lin <i>et al.</i> 2011)
OST1/SnRK2.6	-	Cytoplasm	(Wang <i>et al.</i> 2015)
PAL	-	Cytoplasm	(Vanzo <i>et al.</i> 2014)
Trx	ND	Cytoplasm	(Lin <i>et al.</i> 2012)
GR	+/=	Cytoplasm & Chloroplast	(Bai <i>et al.</i> 2011; Begara-Morales <i>et al.</i> 2012)
Rubisco	-	Chloroplast	(Abat & Deswal 2009)
PrxII F	-	Mitochondria	(Camejo <i>et al.</i> 2013)
CAT	-	Peroxisome	(Ortega-Galisteo <i>et al.</i> 2010)
DHAR	+	Peroxisome	(Bai <i>et al.</i> 2011)
GOX	-	Peroxisome	(Ortega-Galisteo <i>et al.</i> 2010)
MDAR	-	Peroxisome	(Begara-Morales <i>et al.</i> 2012)
MDH	-	Peroxisome	(Ortega-Galisteo <i>et al.</i> 2010)

**Key: ND, Not Detected; +, increased; -, decreased; =, not changed.**

## Figure Legends

**Figure 1. Regulation of S-nitrosylation.** S-nitrosylation is the attachment of an NO group to a cysteine (Cys) thiol (SH) to form an S-nitrosothiol (SNO). The SNO bond is reversible due to its redox sensitivity: i.e. ascorbate, reduced metal ions and GSH can reduce SNO to SH. S-nitrosoglutathione (GSNO), which acts as a stable NO reservoir, can transfer NO to SH group via transnitrosylation. Turnover of GSNO occurs via the enzyme GSNO reductase (GSNOR), generating oxidized glutathione (GSSG) and ammonia (NH<sub>3</sub>).

**Figure 2. Regulation of ascorbate-glutathione cycle by S-nitrosylation during salt stress.** During salt stress the ascorbate–glutathione cycle is modulated via S-nitrosylation of APX, MDAR and GR proteins. MDAR activity is reduced after S-nitrosylation while GR activity is not significantly affected by this modification. However, APX activity is enhanced by S-nitrosylation. This regulation suggests that APX works to detoxify hydrogen peroxide while GR supports the regeneration of GSH, thereby maintaining the antioxidant capability of the ascorbate-glutathione cycle under stress conditions. APX, ascorbate peroxidase; GR, Glutathione reductase; MDAR, monodehydroascorbate reductase.

**Figure 3. Model showing the role of NO and S-nitrosylation during drought stress signalling.** Drought stress triggers ABA accumulation which in turn can induce the production of H<sub>2</sub>O<sub>2</sub> leading to the generation of NO, which results in stomatal closure via the activation of a MAPK pathway. On the other hand, OST1/SnRK2.6 is a central component that regulates ABA signalling via a phosphorylation cascade. NO can regulate ABA signaling through S-nitrosylation of OST1/SnRK2.6, which inhibits its kinase activity, thus acting as a negative regulator of ABA signalling.

**Figure 4. S-nitrosylation regulates different nodes of abiotic stress signalling.** During abiotic stress, proteins related to different nodes of stress signalling become targets for S-nitrosylation/denitrosylation. These proteins include antioxidant proteins, proteins involved in RNS/ROS, cellular metabolism and also proteins that regulate programmed cell death (PCD). These proteins change their S-nitrosylation pattern during stress to control cell fate in two ways: either

stress acclimation or PCD. Rubisco, ribulose-1,5-bisphosphate carboxylase/oxygenase; GAPDH, glyceraldehyde 3-phosphate dehydrogenase; Trx, thioredoxin; AHb1, haemoglobin 1; APX, ascorbate peroxidase; GR, Glutathione reductase; DHAR, dehydroascorbate reductase; GOX, glycolate oxidase; PrxII F, peroxiredoxin II F; MDAR, monodehydroascorbate reductase; CAT, catalase.

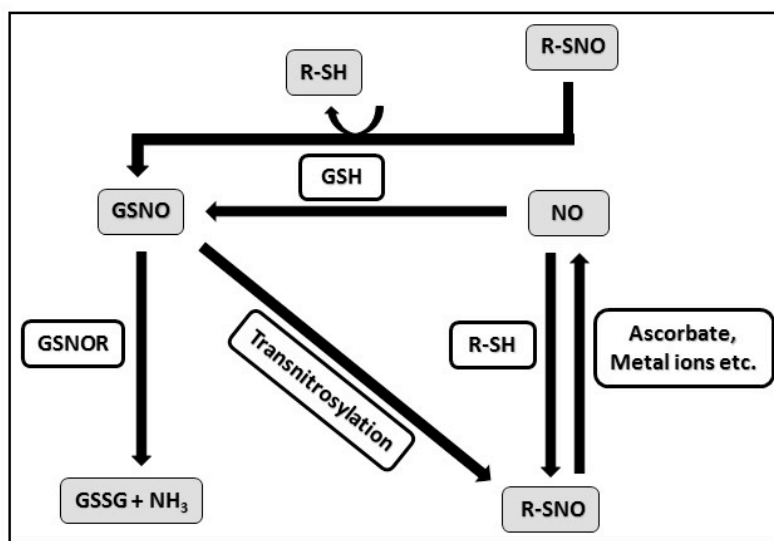


Figure 1. Regulation of S-nitrosylation.

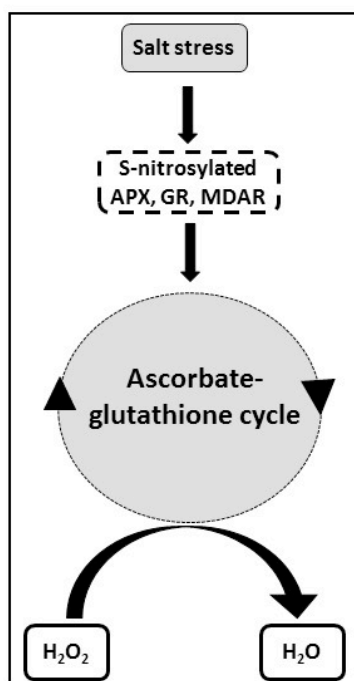


Figure 2. Regulation of ascorbate-glutathione cycle by S-nitrosylation during salt stress.

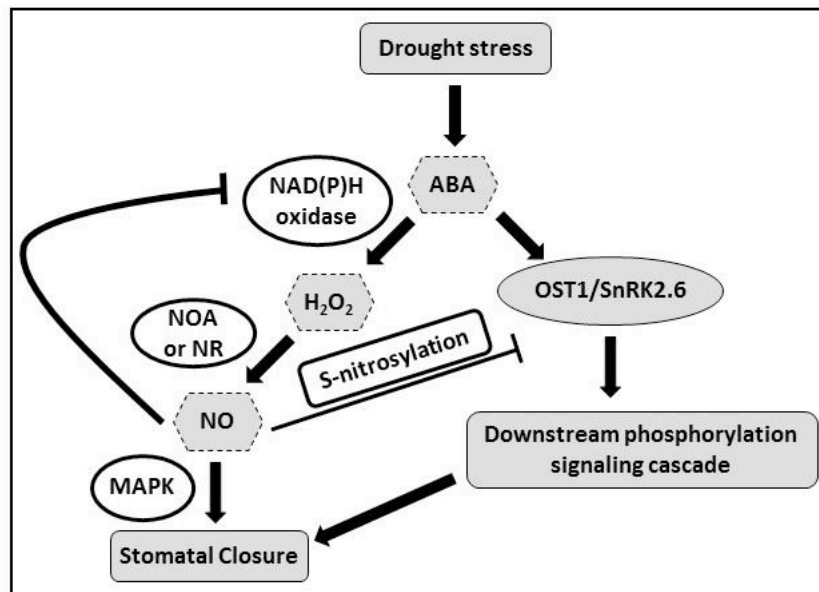


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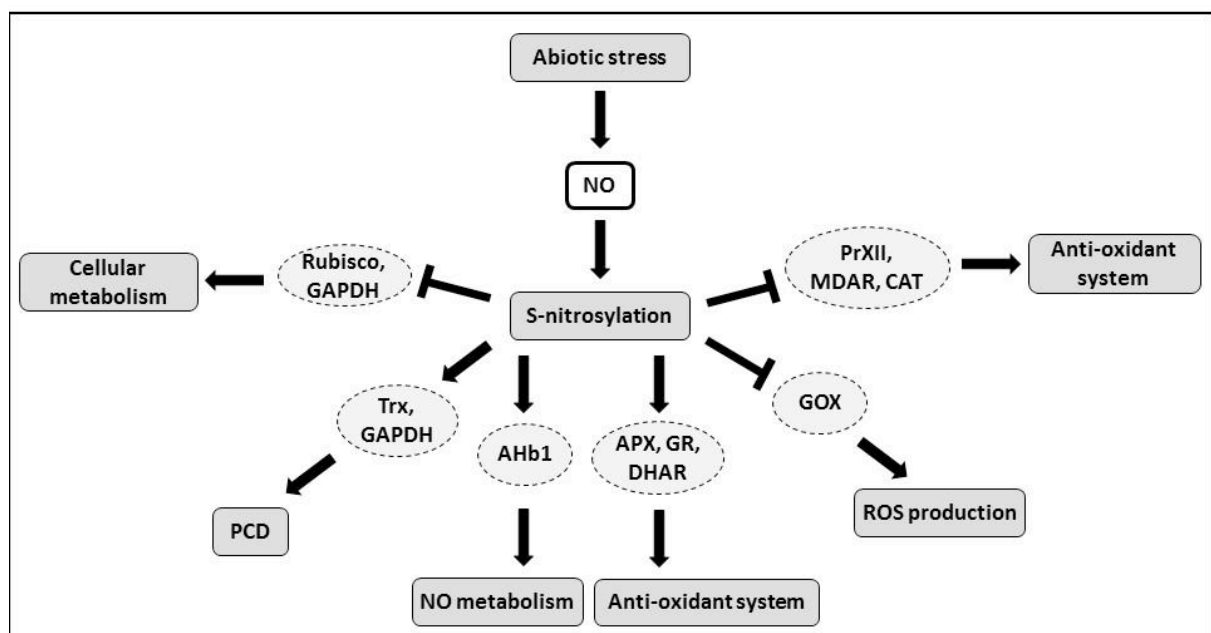


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